thromboplastin time (APTT) were normal. She was given intravenous cefoperazone and 15 more vials of ASV.

Two hours later, her GCS improved and she passed 80 ml of urine. Her Parotid glands on both sides were now swollen (***Fig.* 1**) and tender with normal overlying skin. Her clinical status worsened in next 2 hours, and she developed hypotension and muffling of heart sounds. She was put on inotropes and other supportive care but could not be revived.

The clinical manifestations of viper bite vary from minor local symptoms to life threatening hepatotoxic or vasculotoxic features. Bilateral parotid enlargement is mentioned as one of the clinical signs of viperine envenomation in the Indian National Snakebite Protocol 2009 [1]. However, this is a rare clinical sign, and has been only occasionally reported in adult victims [2,3]. The cause of parotid swelling in viper bite is unknown, but it seems to represent a poor prognostic outcome [4].

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FIG.1 *Parotid swelling in the patient of viper envenomation. (See color image at website)*

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Esophageal Stricture Following Retained Foreign Body in a Child

Infants put almost everything into their mouths and toddlers eat just about anything. The majority of foreign body ingestions occur in children between the ages of six months and three years [1]. Only 10 to 20 percent of foreign bodies require endoscopic removal, and less than 1 percent require surgical intervention [1,2].

Retained foreign body in esophagus is very rare presentation, which may damage the mucosa leading to stricture or fistula. We report a case of 8-yr-old male child who was brought to medical attention with complaints of vomiting after meals and difficulty in swallowing food for the past 4 years, along with cough and noisy breathing for three months. There was a history of ingestion of a 2-rupee coin prior to start of the symptoms, passage of which the parents never noticed subsequently in stools, and they did not seek any further medical attention. After admission, *X*-ray chest was done which revealed a radio-opaque shadow in the mid esophagus; lungs were normal (***Fig.* 1**). Upper gastrointestinal endoscopy revealed a stricture at 12 cm from incisors; proximal esophagus showed diverticulum and the coin was seen distal to the stricture. The stricture was dilated using Savory Gillard dilators and the coin was pushed distally into the stomach. (***Fig* 2**). A contrast *X*-ray swallow (gastrografin) study was normal. After one week, the child passed the coin in the stool. Repeat dilatation was done after 7 days and 21 days. After dilatation, the child started accepting feeds orally without any complaints; there was no requirement of dilatation after three initial sessions.

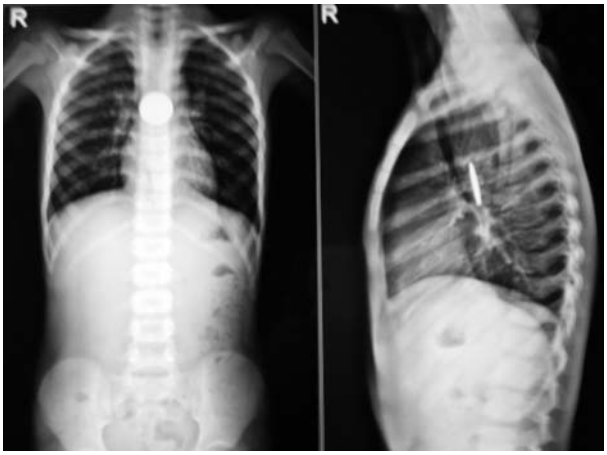

FIG. 1 *X-ray chest PA and Lateral view showing radio-opaque foreign body.*

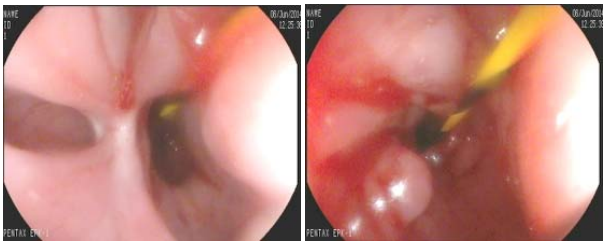

FIG. 2 *Stricture and diverticulum in esophagus.*

Retained esophageal foreign bodies are uncommon in pediatric practice and they should be endoscopically removed as soon as possible. In our patient, the appropriate management for coin ingestion was not done at the time of ingestion and thus led to retained foreign body and stricture formation. Esophageal stricture resulting from a long-standing lodgment of metallic foreign bodies has been reported earlier [3,4]. As retained esophageal foreign body can lead to stricture, a timely appropriate management should be done at the time of ingestion.

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Pamidronate for Long-term Control of Hypercalcemia Associated With Williams Syndrome

Hypercalcemia in Williams Syndrome is usually mild and transient, but in about 5% of patients, it may be severe, and associated with medullary nephro-calcinosis [1].

A 3-year-old boy, second born of non-consanguineous parents with uneventful perinatal history, presented to us with global developmental delay and failure to thrive. He had history of feeding difficulty, constipation, increased frequency of micturition, and increased irritability, and failure to thrive. Examination revealed facial features characteristic of Williams syndrome. Initial blood investigations revealed total serum calcium of 14.5 mg/dL and phosphorus of 6.2 mg/dL. Serum 25-OH vitamin D was 21.4 ng/mL, serum i-PTH was 2.5 pg/mL and urinary calcium creatinine (Ca/Cr) ratio was 2.4. Repeat values were consistent with PTH-independent hypercalcemia and hypercalciuria. Ultrasonography of kidneys showed bilateral dense medullary nephrocalcinosis. Genetic analysis using Fluorescence in situ hybridization (FISH) was done which confirmed deletion in region of 7q11.23.

We started the child on calcium-restricted diet, intravenous fluids for hydration, and furosemide to reduce serum calcium levels. Despite these measures, there was no decrease in serum calcium levels for 48 hours and the repeat serum calcium level was 15.2mg/dL. We administered single dose of pamidronate (1mg/kg) as intravenous infusion over 6 hours. Gradually the serum calcium levels decreased over a period of 3 days to 10.2 mg/dL. On subsequent follow up visits, at 2,4, 8, 12 weeks, and 6 and 12 months, the serum calcium level and urinary calcium creatinine ratio were in normal range. His irritability, feeding difficulty and constipation resolved, and he was gaining weight.

Though the association of Williams syndrom with hypercalcemia is well established, the exact mechanism